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ELECTROPHYSIOLOGIC STUDIES

Transvenous Catheter Ablation of Extranodal Accessory Pathways

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Twelve patients with an accessory pathway and recurrent symptomatic reciprocating tachycardia or atrial fibrillation, or both, underwent attempted transvenous catheter ablation of the accessory pathway. In one patient with a small right coronary artery, the pathway was along the right free wall. In 11 patients, the pathway was located at or within 15 mm of the coronary sinus os. For these patients, a quadripolar electrode catheter was placed in the coronary sinus and positioned, if possible, so that the proximal pair of electrodes straddled the pathway. For those patients with a pathway >5 mm within the coronary sinus, the most proximal electrode was placed at the os. This proximal pair of electrodes was connected to the cathodal output of a defibrillator with an anterior chest wall patch serving as the current sink. Two shocks were then delivered for a cumulative energy of 500 to 600 J (stored energy).

Among the eight patients with a pathway at or within 5 mm of the coronary sinus os, conduction over the pathway was abolished in five and modified in one. Among the four patients with a pathway farther from the os (10 to 15 mm) and along the right free wall, pathway conduction was modified only in two. Rupture of the coronary sinus did not occur in any patient. There were no serious complications. Minor damage surrounding the area of ablation was seen at the time of surgical division of the accessory pathway in two of five patients with unsuccessful ablation who subsequently underwent surgery.

Transvenous catheter ablation of accessory pathways appears to be a safe and reasonably effective alternative to surgery for those patients with a pathway at or near the coronary sinus os, but not for those with a pathway farther from the septum. Although subsequent intraoperative mapping was more difficult because of the loss of large, discrete atrial electrograms, surgical division was successful in all five patients after unsuccessful catheter ablation.

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The success of transvenous catheter ablation using high energy electrical discharges of the atrioventricular (AV) junction (1–5) was followed by its application to accessory pathways. Several reports (6–15) have described catheter ablation of accessory pathways, primarily in the posteroseptal area but also along both the left and the right free wall.

The purpose of this report is to describe our experience with ablation of extranodal accessory pathways. We were particularly interested in defining those variables that influenced the success of the procedure, the gross appearance of the ablated material, the effect of the procedure on the electrical properties of the ablated tissue, the mechanism of action of the ablation and any complications of the procedure, including any difficulty it may engender in the subsequent surgical division of an accessory pathway in patients in whom ablation was unsuccessful.

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Methods

Study patients. Eight men and four women were referred for electrophysiologic evaluation and treatment because of symptomatic, recurrent, drug-refractory supraventricular tachyarrhythmias. Their mean age was 36 ± 10 years (mean ± SD) and the mean duration of their symptoms of tachycardia was 13.9 years (range 1 to 30). Six patients had previously presented with episodes of orthodromic AV reciprocating tachycardia only, four with both reciprocating tachycardia and atrial fibrillation (with ventricular responses of 220 to 300 beats/min) and two with atrial fibrillation only. The patients had previously been unsuccessfully treated with or proved intolerant to a mean of 1.8 ± 0.9 antiarrhythmic drugs.

Baseline electrophysiologic study. Each patient underwent a baseline electrophysiologic study at least five half-lives after the discontinuation of antiarrhythmic drugs. Four of

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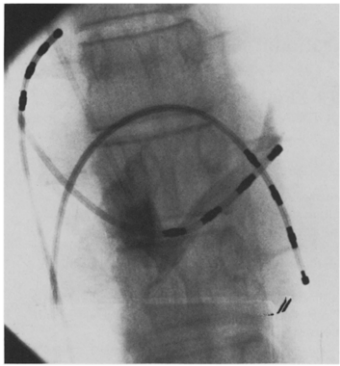

Figure 1. Quadripolar electrode catheters are located in the high right atrium and the right ventricular apex. A quadripolar catheter with a central lumen is placed in the coronary sinus. Contrast material fills the lumen of the coronary sinus and spills into the right atrium. The atrial insertion point of the accessory pathway relative to the coronary sinus os is precisely determined.

quadripolar electrode catheters (USCI) were inserted into the femoral or right internal jugular or left subclavian vein and positioned in the high right atrium, coronary sinus, right ventricular apex and across the tricuspid anulus to record a His bundle electrogram. Baseline conduction properties of the accessory pathway and AV node were determined. Orthodromic AV reciprocating tachycardia was induced in all patients except Patient 7, whose clinical arrhythmia was atrial fibrillation with pre-excitation.

The atrial insertion site of the accessory pathway was mapped by recording both bipolar and unipolar electrograms (bandpass filter settings 25 to 500 Hz) throughout the coronary sinus and around the tricuspid anulus during orthodromic reciprocating tachycardia and rapid ventricular pacing. In those 11 patients with earliest retrograde atrial activation recorded near the coronary sinus os, a 6F electrode catheter with a central lumen was positioned in the coronary sinus. Contrast material was then injected through the lumen to outline the coronary sinus and its os and, by making use of the known interelectrode distance, the location of the accessory pathway relative to the coronary sinus os was determined within 5 mm (Fig. 1).

Catheter electrical ablation. Patients were considered candidates for ablation if the pathway was located within 15 mm of the coronary sinus os (11 patients) or if the pathway was along the right free wall, but not adjacent to the right coronary artery (1 patient). After a discussion of the options available, including treatment with other antiarrhythmic drugs, surgical accessory pathway division or catheter ablation, the patients provided informed consent under a protocol approved by the Institutional Review Board of Sequoia Hospital, Redwood City, California.

Patients were brought to an operating room equipped with cineangiographic apparatus; a cardiac surgeon experienced in performing electrophysiologic surgery was immediately available. Electrode catheters were placed in the right atrium and right ventricular apex through a femoral vein. For those 11 ablations near the coronary sinus os, an electrode catheter with a central lumen was inserted into the left subclavian vein and positioned in the coronary sinus. A contrast injection was once again utilized to localize the accessory pathway relative to the coronary sinus os. A 6F quadripolar electrode catheter with 1 cm interelectrode distance (USCI) was then placed in the coronary sinus. For patients whose earliest retrograde atrial activation was within 10 mm of the coronary sinus os, the catheter was positioned so that, during orthodromic reciprocating tachycardia, unipolar electrograms recorded from the proximal pair of electrodes demonstrated equal and early ventriculoatrial (VA) conduction times (Fig. 2). For patients with a pathway located ≥10 mm within the coronary sinus, care was taken not to place the most proximal electrode within the os. For these patients, the third electrode of the quadripolar catheter could be as far as 5 mm away from the earliest recorded retrograde atrial activity. Before the ablation, the atrial (coronary sinus) placing threshold obtained using these proximal electrodes was determined as a measure of electrode-tissue contact.

The patients were then anesthetized with sodium thiopental and intubated. Generalized paralysis was obtained by the infusion of succinylcholine. The proximal pair of electrodes was made electrically common and connected to the cathodal output of a defibrillator (Datascope Corp.) that had previously been shown to deliver a dampened sinusoidal waveform of 35 ms duration. A 16 cm patch electrode (R-2 Corp.) was placed over the left anterior chest and a 300 J (stored energy) discharge, synchronized to the QRS complex, was delivered. After 20 min a second 200 or 300 J shock was delivered using a new catheter. In Patient 2, a separate ablation procedure was done 3 days after the first unsuccessful ablation utilizing an electrode patch placed below the left scapula.

The catheter ablation protocol for the single patient (Case 12) with a right free wall accessory pathway differed in that a 6F quadripolar catheter was introduced into the right internal jugular vein. The patient had previously been shown to have a small, nondominant right coronary artery. The distal electrode was positioned at the tricuspid anulus, previously outlined by right ventriculography, and adjusted

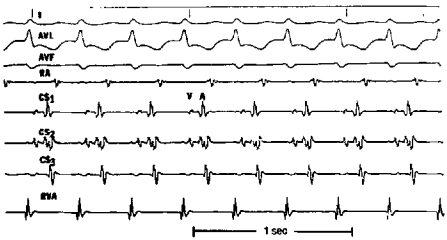

Figure 2. Electrograms during positioning of the quadripolar electrode catheter immediately before the ablation during reciprocating tachycardia. The catheter is placed such that the third and fourth electrodes record equally early unipolar atrial electrograms. CS_1, CS_2 and CS_3 = unipolar recordings from fourth, third and second poles of the coronary sinus catheter, respectively; RA = right atrium; RVA = right ventricular apex.

to lie over the accessory pathway as determined by VA conduction times during reciprocating tachycardia. After the distal electrode was connected to the cathodal output of the defibrillator, a 200 J discharge was delivered. This was followed 20 min later by a 150 J shock delivered without exchanging the catheter.

After the ablation, the patients were continuously monitored for 5 days and serial serum creatinine kinase and creatine kinase-MB fraction levels determined.

Repeat electrophysiologic study. All patients underwent repeat electrophysiologic testing 4 days to 1 month after the ablation. The presence or absence of an accessory pathway and the conduction characteristics of the accessory pathway or AV node, or both, were determined.

Surgery. Five (Cases 2, 7, 8, 10 and 12) of the six patients with unsuccessful catheter ablation underwent surgical division of the accessory pathway. Particular attention was paid to any evidence of damage from the ablation attempt, the relation of the coronary sinus to the anulus near the septum and the nature of the electrograms at or near the ablation attempt.

Results

Baseline electrophysiologic study (Table 1). An orthodromic reciprocating tachycardia was induced in all patients except Patient 7, who had clinically demonstrated only atrial fibrillation with pre-excitation. The cycle length of the tachycardia ranged from 270 to 345 ms (mean 292). The earliest atrial activity occurred 65 to 120 ms after the onset of the ventricular electrogram. In four patients, the earliest retrograde atrial activity was at the coronary sinus os; it occurred 5 mm within the os in two patients, 10 mm within the os in two patients, and 15 mm within the os in one patient. One patient (Case 10) had two distinct pathways, one at the os and one 15 mm within the os. In one patient, the earliest retrograde activity was seen 5 mm outside the os on the posterior right atrial wall. One patient (Case 12) had a single

Table 1. Baseline Conduction Characteristics of the Accessory Pathway in 12 Patients

Patient No.	Site of Pathway	Anterograde (ms)		Retrograde (ms)		Tachycardia Cycle Length (ms)	VACT (ms)
		1:1 Conduction	ERP	1:1 Conduction	ERP		
1	CS os	240	250	250	210	280	70
2	CS os	CBT	CBT	280	270	340	110
3	CS os	340	330	280	250	270	65
4	CS os	325	325	310	290	295	120
5	5 mm in os	220	250	240	230	290	85
6	5 mm in os	210	220	270	270	295	105
7	10 mm in os	260	280	270	260	—	85
8	10 mm in os	CBT	CBT	260	250	250	80
9	15 mm in os	230	<230	230	<220	275	65
10*	CS os and 15 mm in os	250	260	260	240	345	95
11	5 mm outside os	230	220	250	240	290	85
12	Right free wall	210	220	300	210	280	70

*Two discrete pathways were identified, one located at the os and one located 15 mm within the os. CBT = concealed bypass tract; CS = coronary sinus; ERP = effective refractory period; VACT = ventriculoatrial conduction time.

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Table 2. Results of the Ablation in 12 Patients

Patient No.	Energy* (joules)	Accessory Pathway Conduction: Immediate Results‡	Accessory Pathway Conduction: Delayed Results (7 to 30 days)
1	300/200	0	0
2†	300/300	0	+
3	300/200	0	0
4	300/200	+	+
5	300/300	0	0
6	300/300	0	0
7	300/300	+	+
8	300/300	+	+
9	300/200	0	Modified
10†	300/300	+/0	+/0
11	300/200	0	Modified
12	200/150	0	Modified

*Listed are the stored energies (joules) used for the first and second shocks; †Patient 2 underwent two ablations with similar immediate and delayed results at the same energy intensity; ‡conduction over the pathway located at the os was abolished. 0 = no conduction over accessory pathway; + = conduction over accessory pathway.

right free wall pathway 3 cm to the right of the septum and with a small, nondominant right coronary artery.

Immediate ablation results (Table 2). No tachyarrhythmias or hemodynamic deterioration was seen. Atrial asystole occurred in all patients (mean duration 8 ± 8 s). In addition, all patients except Patient 12 developed AV block requiring ventricular pacing (mean duration 53 ± 33 s); this uniformly resolved within 2 min.

Conduction over the accessory pathway was abolished initially in all patients. In five patients, conduction returned 3 to 15 min after the first shock. After the second shock, accessory pathway conduction returned in four of these five patients within 5 to 12 min. In Patient 12, after an additional 150 J shock, no conduction was seen after 20 min.

Repeat electrophysiologic studies (Table 3). After 4 to 30 days, electrophysiologic studies were repeated. In four patients (Cases 1, 3, 5 and 6) there was no evidence of conduction over the accessory pathway. These patients have remained asymptomatic without drugs and with no electrocardiographic (ECG) evidence of pre-excitation over a mean period of 16 ± 6 months.

In Patient 10, conduction over the pathway located at the os was abolished as assessed by retrograde atrial activation during tachycardia as well as during ventricular pacing whereas the pathway located 15 mm within the os was unaffected. The anterograde pre-excitation pattern on 12 lead ECG also suggested loss of anterograde function of the more proximal pathway. Because the tachycardia appeared to be unaffected, surgical division of the pathway was undertaken. Loss of conduction over the pathway at the os was confirmed during surgical mapping and the pathway located well within the coronary sinus was successfully divided.

In two patients (Cases 9 and 11) conduction over the accessory pathway appeared abolished at the end of the procedure but returned after 48 h. However, the conduction was found to be significantly modified. In Patient 9, conduction over the accessory pathway was abolished by procainamide hydrochloride (Pronestyl) (level 4.2 μg/ml), which previously had had no effect either clinically or at the time of initial study. Patient 9 remains asymptomatic with this drug after 18 months. The accessory pathway in Patient 12 was also significantly modified (Table 3) but, because he desired a complete cure, surgical division of the accessory pathway was subsequently performed.

In Patient 11, anterograde conduction over the pathway was abolished. Retrograde conduction was modified and the induced reciprocating tachycardia was considerably slower. He remains free of symptoms, without drug therapy, after 5 months. In four patients (Cases 2, 4, 7 and 8), conduction over the accessory pathway and the induction of tachycardia appeared to be unchanged by the ablation.

Surgical findings. Five patients (Cases 2, 7, 8, 10 and 12) with unsuccessful ablation underwent surgical division of the accessory pathway using a standard surgical approach

Table 3. Repeat Electrophysiologic Study After Unsuccessful Ablation in Eight Patients

Patient No.	Anterograde (ms): 1:1 Conduction	Anterograde (ms): ERP	Anterograde (ms): 1:1 Conduction After Isoproterenol	Retrograde (ms): 1:1 Conduction	Retrograde (ms): ERP	Tachycardia Cycle Length (ms)
2	CBT	CBT	CBT	290	270	380
4	280	270	—	250	250	315
7	280	280	—	260	260	—
8	CBT	CBT	CBT	250	240	265
9*	330	330	320	340	330	350
10	260	<250	—	270	<250	350
11†	CBT	CBT	CBT	310	320	345
12	340	330	310	330	310	350

*Pathway conduction was abolished with Pronestyl, which had had no effect on the pathway before ablation; †anterograde pathway conduction was abolished and retrograde conduction was modified. Abbreviations as in Table 1.

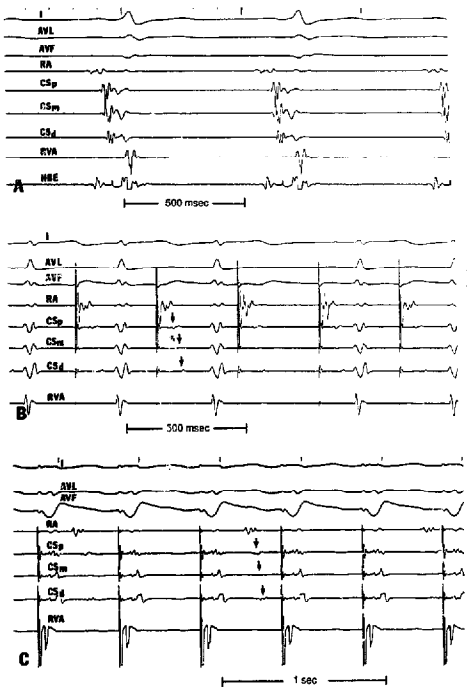

Figure 3. Effect of ablation on local atrial electrograms (bipolar recordings). As compared with those before the ablation (**panel A**), the atrial electrograms after ablation (**arrows** in panels **B** and **C**) are considerably smaller, indistinct and somewhat broadened. During right atrial pacing (**B**) and ventricular pacing (**C**), there is no accessory pathway conduction. CSd = distal coronary sinus; CSm = midcoronary sinus; CSp = proximal coronary sinus; HBE = His bundle electrogram. Other abbreviations as in Figure 2.

through a right atriotomy 4 to 60 days after the ablation. Visible damage from the ablation was seen in two patients. In Patient 8 there was a 5 mm area of whitish discoloration on the epicardium but no visible damage endocardially. Patient 12, who had a right free wall pathway, was found to have a small hemorrhagic pericardial effusion and a 10 mm circular ecchymosis epicardially above the AV groove and a punctate red lesion above the tricuspid anulus endocardially. Surgical division of the pathway was successful in all five patients.

Other effects from the ablation. Minor complications were seen in Patient 12, who developed a brief episode of pericarditis-like chest discomfort 24 h after the ablation, and in Patient 5, who had an ectopic ventricular triplet 36 h after ablation. Atrial arrhythmias were not seen in any patient. The mean creatine kinase increase was 673 ± 524 IU/liter with an MB fraction of 24 ± 25 IU/liter.

Effect on atrial electrograms. The atrial recordings at the ablation site measured after ablation indicated a reduction in amplitude resulting in an atrial electrogram amplitude of 0.22 ± 0.20 (range 0.12 ± 0.60) of the amplitude before ablation. In addition, the signals were broadened and were frequently indiscrete, suggesting local conduction delay; this effect persisted with time (Fig. 3).

Because of these changes, interpretation of subsequent activation sequence mapping was sometimes confusing. For example, Patient 8 underwent detailed mapping at repeat electrophysiologic study. At the end of the ablation proce-

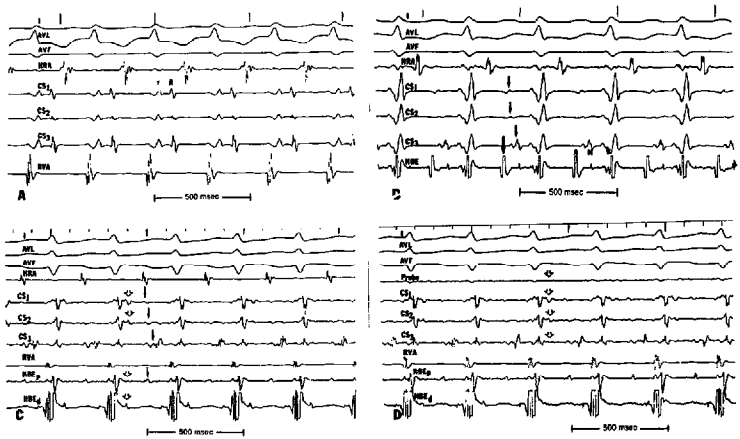

Figure 4. Patient 8. Before the ablation (**panel A**), a single accessory pathway was identified 10 mm within the coronary sinus (CS) os, as shown by ventriculo-atrial (VA) conduction times during tachycardia recorded (unipolar) from electrodes 4, 3 and 2 (designated CS_1, CS_2 and CS_3, respectively). Immediately after the ablation (**panel B**), it initially appeared that the posteroseptal pathway was successfully ablated, but that a second pathway was conducting retrogradely to sustain a tachycardia. Atrial electrograms recorded from the high right atrium (HRA) are well before those from the coronary sinus (CS, CS_2, CS_3) (**small arrows**) or the septum (**large arrow**). However, because the anterograde pre-excitation pattern appeared unchanged, a complete map during tachycardia was done (**panel C**) showing that the posteroseptal pathway actually remained (**open arrows**) but that local atrial conduction was dramatically altered giving the misleading map seen in panel B. A catheter placed against the septum near the os ("probe" in panel D) confirms the alteration in local atrial electrograms. HBE_d = distal His bundle electrogram; HBE_p = proximal His bundle electrogram. Other abbreviations as in Figure 3.

dure, it appeared that the posteroseptal accessory pathway was successfully ablated but that a latent right free wall pathway was uncovered. Prompted by the fact that the anterograde pre-excitation pattern was unchanged, complete mapping revealed that the posteroseptal pathway actually remained, but that atrial conduction in the vicinity had been grossly altered (Fig. 4).

Despite relatively normal-appearing atrial tissue, the alteration in atrial electrograms was immediately obvious at surgical mapping (Fig. 5). Because of this, it was sometimes difficult to precisely localize the atrial insertion site of the accessory pathway. Nevertheless, surgical division of the accessory pathway was successful in all patients.

Atrioventricular node conduction (Table 4). All patients underwent an electrophysiologic study during which AV node conduction, in the absence of an accessory pathway, could be assessed either after a successful ablation or postoperatively. All patients exhibited anterograde conduction. There has been no evidence of AV node anterograde block during long-term follow-up.

Factors affecting the success of the procedure. The factors examined included the position of the accessory pathway, the rapidity with which the pathway was capable of conducting, the contact of the ablating electrodes with atrial tissue, the relation of the coronary sinus to the anulus and the presence of a concealed bypass tract. All pacing thresholds using the ablating electrodes were <2 mA, suggesting adequate electrode-tissue contact. For the five successful ablations, the three ablations that modified conduction and the four unsuccessful ablations, the mean cycle length at which the pathway sustained 1:1 conduction before ablation was 270, 210 and 268 ms, respectively (p = NS). The single factor that clearly affected the success of the ablation was the relation of the pathway to the coronary sinus os. For the eight patients with a pathway at or within 5 mm of the os, the success rate was 63%. For the three patients with a pathway

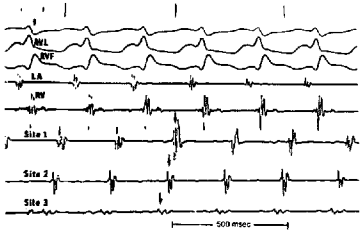

Figure 5. Patient 2. Epicardial surgical map of accessory pathway during tachycardia after unsuccessful catheter ablation. As the probes on the atrial side of the atrioventricular groove are moved by 1 cm steps (sites 1 and 2) toward the area of the septum (site 3) from the right free wall, the atrial electrograms become smaller, less distinct and broadened. LA = left atrial appendage; RV = right ventricle.

10 to 15 mm from the os, only one pathway was modified. In addition, the second pathway located 15 mm within the os in Patient 11 was not affected by the ablation.

Discussion

The results in this series of 11 patients suggest that transvenous catheter ablation is safe and reasonably effective (63%) for carefully selected patients with an accessory pathway at or near the coronary sinus os. The success rate for pathways farther from the os is considerably less. In addition, a price is paid in the loss of large, discrete atrial electrograms, which may confuse future mapping studies and make surgery more difficult.

Table 4. Late Atrioventricular Node Conduction in 12 Patients

Patient No.	Anterograde (ms)		Retrograde (ms)	
	1:1 Conduction	Isoproterenol	1:1 Conduction	Isoproterenol
1	370	250	VAD	VAD
2	560	350	VAD	VAD
3	350	290	VAD	VAD
4	310	250	270	<260
5	310	270	VAD	VAD
6	320	280	290	—
7	420	370	510	—
8	410	240	250	<240
9	340	—	460	—
10	530	330	VAD	VAD
11	330	260	—	—
12	310	250	270	<260

VAD = ventriculoatrial dissociation.

Risk of coronary sinus rupture. The fact that coronary sinus rupture was not seen in this series is likely due to the relatively "unaggressive" protocol used. Most importantly, discharges were never delivered within the coronary sinus and the relation of the pathway and the os was precisely defined. In previous reports of rupture during ablation attempts (10,16,17), the discharges were delivered within the confines of this thin-walled venous structure, with rupture probably resulting from the barotraumatic effects of the discharge. In addition, we restricted the number of discharges to two, and used a new catheter for the second shock to obviate any potential problems with insulation breakdown from the first discharge and subsequent misdirection of the energy to a more distal electrode (18,19). The shocks were cathodal, which are less barotraumatic than are anodal shocks (20). Finally, generalized paralysis was induced in patients before delivery of the discharge to avoid the violent motion of the heart caused by contraction of the diaphragm with the shock and the possibility of direct puncture of the coronary sinus with the tip of the ablating catheter. However, we emphasize that, although there were no major complications, the procedure, which we consider investigational, was performed in the operating room with a cardiac surgeon available to correct any complication, and to complete the division of the pathway surgically.

Success rate. Whether the use of more discharges or of discharges deeper within the coronary sinus would increase the success rate of the procedure is unknown. It is of interest that in those patients with an ultimately successful ablation, the accessory pathway appeared to be blocked with the first shock. The results in this small number of patients suggest that a second discharge may be unnecessary. Certainly the second ablation attempt in Patient 2 (for a cumulative dose of 1,200 J) was no more effective than the first. Because of prior reports of rupture of the coronary sinus when energy was delivered within the os (10,16,17), no shocks were delivered within the coronary sinus even in those four patients with a pathway located 10 to 15 mm within the os. It was hoped that the destructive energy might diffuse through atrial tissue sufficiently to interrupt pathway conduction. However, in view of the low success rate of this approach (one of four pathways modified), we have since abandoned ablation attempts using high energy discharges of pathways that are >5 mm within the os. The protocol used appears to provide an acceptable compromise between efficacy and safety.

Effect on atrial tissue. The alteration of local atrial electrograms near the site of energy delivery is notable. The electrical activity of atrial tissue in the vicinity of the ablation is so dramatically affected that it appears that the mechanism of successful ablation may be disruption either of the accessory pathway or of atrial tissue surrounding the atrial insertion point. This latter possibility is further suggested by the fact that because of interpatient variability in the anatomic site of the coronary sinus os relative to the

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anulus fibrosis, in some patients the os may be removed a considerable distance from the point of actual AV continuity. In unsuccessful ablations, the disruption of atrial conduction and the resultant fragmented, small atrial signals sometimes complicated interpretation of subsequent electrophysiologic studies (Patient 8). In patients with an unsuccessful ablation, surgical mapping was uniformly more difficult, although surgery was successful in all patients.

In contrast to canine studies with AV node-His bundle axis ablations (1,2,21) and with atrial (22) and ventricular ablations (23–25), the gross damage seen at the time of surgery was relatively minor. There was no obvious effect on the coronary artery, and ECGs obtained after ablation suggested that coronary artery spasm did not occur. The effect of the ablation on the electrical activity of the atrium was clearly greater than the gross anatomic effect.

Transient sinus node arrest and AV block were observed in all of the patients. Nevertheless, subsequent sinus node function and AV conduction appeared to be normal, although a direct comparison of AV node function before and after ablation was not possible because of the presence of accessory pathway conduction before the ablation. Histologic studies of posteroseptal ablations in a canine model have not shown damage in the AV node-His bundle axis (16). The mechanism of this temporary disruption in conduction is not clear but may be related to either barotrauma (16,20) or the effect of massive voltage changes causing a derangement in transmembrane gradients at the cellular level (26,27).

Role of location of coronary sinus os relative to accessory pathway. Although various factors that might possibly affect the success of the ablation (including the rapidity of pathway conduction, the presence of a concealed bypass tract and the relation of the os relative to the pathway) were analyzed, only the location of the os relative to the pathway was important. This is not surprising because discharges were delivered at or just inside the coronary sinus os. It is possible that a major factor affecting the success of the ablation is the distance of the os to the actual point of AV continuity such that tissue destruction surrounding the os may not extend to the anulus fibrosis. Although there was no obvious difference in the distance of the os to the anulus in those patients who underwent surgery, the number of patients was too small to arrive at a definite conclusion. When the distance of the ablating catheter from the anulus was estimated by a comparison of the amplitudes of the atrial and ventricular electrograms recorded at the os before the ablation (22), no significant difference was noted between successful and unsuccessful ablations.

Limitations of study. There were several limitations to the study. Follow-up coronary angiograms were not obtained for any patient. On the other hand, there was no gross evidence of damage at the time of surgery near the coronary artery in any patient except Patient 12, in whom, if the right coronary artery had not been so small, an area of ecchymosis would have been near the artery. In addition, immediate and late ECGs did not show any evidence of ventricular myocardial injury. Late coronary sinus angiograms were also not obtained. Again, there was no evidence of damage in those patients who subsequently underwent surgery. Repeat, late coronary angiograms are planned in all of these patients. Finally, it is possible that the minimal tissue damage seen at surgery was more pronounced in those patients with successful ablation. This seems unlikely because the atrial electrograms were dramatically altered in all patients.

Technique. The optimal method of ablating an accessory pathway is not known. In addition to the obvious issues regarding the amount of energy to be used, the number of discharges delivered and the position of the ablating catheter, other questions remain. We generally used two electrodes of the catheter to disperse the ablating energy, although a more precise delivery with a single electrode may be preferable. Similarly, because of what appears to be a vectorial component to the discharge energy, the ideal area for placement of an anodal patch is not evident. We chose to place the anode over the left side of the chest with a view toward including the septal area in the vector of ablating energy. Finally, a more precise mode of delivering energy, possibly radiofrequency energy (28), or the use of an energy waveform associated with less barotrauma, may be preferable. In fact, it is possible that, with other forms of energy, ablation of accessory left wall pathways may be feasible.

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